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Abstracts

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Modifications of calciuria and phosphaturia following glucose ingestion in patients with calcium urolithiasis. *J. Aubert, A. Ulmann, B. Lacour, and J. L. Funck-Brentano. Département de Néphrologie, Hôpital Necker, Paris, France.* Increased urinary calcium excretion in patients with calcium urolithiasis could be, at least in part, dependent on carbohydrate intake. To test this hypothesis, we administered to 8 hypercalciuric stone-formers (HCI), 8 normocalciuric stone-formers (NCI), and 7 control subjects a 75-g glucose oral load, after they were fasted overnight. Urine was collected during three 30-min periods (one control period, before glucose ingestion and two experimental periods 30 to 60 and 60 to 90 min after glucose ingestion). High urine output was obtained by infusing physiologic saline. Plasma samples were collected at the middle of each period. Calcium, phosphorus, creatinine and sodium were measured in each plasma and urine sample. In some patients, plasma $1,25(OH)_2D$ was assayed before and after glucose ingestion. Results obtained after glucose ingestion are as follows: *(1)* In patients with HCI, both Cl_{Ca}/Cl_{Na} and Cl_{Ca}/Cl_{Creat} significantly increased ($P < 0.01$). In patients with NCI, Cl_{Ca}/Cl_{Creat} but not Cl_{Ca}/Cl_{Na} increased significantly ($P < 0.01$). In control subjects, neither Cl_{Ca}/Cl_{Creat} nor Cl_{Ca}/Cl_{Na} increased. *(2)* Although plasma phosphorus significantly decreased in the three groups, TmP/GFR decreased in patients with HCI ($P < 0.05$) or NCI ($P < 0.01$) but not in control subjects. *(3)* In patients with HCI in whom it was assayed, $1,25(OH)_2D$ increased significantly. In conclusion, a glucose load induces hypercalciuria in stone-formers with HCI or NCI and contributes to high plasma $1,25(OH)_2D$ concentrations found in these patients.

Nephrotic syndrome associated with T-cell lymphoma. *D. Belghiti, J. P. Vernant, G. Hirbec, M. C. Gubler, C. André, and A. T. Sobel. Hôpital Henri Mondor, Créteil, France.* It has been known for years that nephrotic syndrome may be associated with malignant lymphomas. In the vast majority of the reported cases, the glomerular disease was of the minimal-change type, and the corresponding hematologic trouble was Hodgkin's disease. In contrast, glomerulopathies associated with non-Hodgkin's lymphomas (NHL) have been rarely observed, and the cell type of the lymphoproliferative disorder was never determined. This is the first case of glomerular disease in a patient with NHL of proven T-cell origin. Data obtained by functional assays, cytochemical staining, and EM were consistent evidence indicating that the lymphoproliferative disorder observed in a 17-year-old black female, with early clinical involvement of liver and spleen and secondary medullar infiltration, was a T-cell derived NHL. The evidence included: E rosette formation with blast cells, absence of surface immunoglobulins and EAC rosettes, positive staining for β-glucuronidase and ultrastructural aspect on EM examination. Nephrotic syndrome appeared shortly after the apparent onset of NHL. The kidney biopsy specimen was examined by light, fluorescence, and electron microscopy. Histologic findings were consistent with the diagnosis of mild focal glomerulosclerosis. The lymphoproliferative disease responded partly and transiently to combined chemotherapy without simultaneous improvement of the nephrotic syndrome. This report supports the concept of an eventual link between T-cell abnormalities and glomerular diseases but more information is obviously needed to assess such a hypothesis.

Effects of nonsteroidal antiinflammatory drugs on the renin release by isolated perfused kidney. *D. Casellas, M. Dupont, and A. Mimran. CHR Saint-Charles, Montpellier, France.* In vivo inhibition of renal prostaglandin (PG) synthesis by nonsteroidal antiinflammatory drugs (NSAID) is associated with a decrease in renin release (RR) in various species including the rat. To elucidate the mechanism(s) of action of such drugs, we assessed the effect of indomethacin (I) and aspirin (AS) on RR by the isolated perfused rat and rabbit kidney. A cell-free medium was perfused at the constant flow rates of 5 and 10 ml/min in rats and rabbits, respectively. Perfusion pressure (PP) and renin concentration (RC) in venous effluent were measured. In addition, NSAID vehicle per se ($NaHCO_3$) had no effect on either RC or PP in control studies; effectiveness of PG synthesis inhibition by the presently used doses of NSAID was checked (radioimmunoassay of PGE_2 in venous effluent). In 7 rats, I was found to have a biphasic effect on RC *(a)* at the dose of 0.55 μM, RC was decreased by 22 $\pm$ 3% and PP slightly increased by 2 $\pm$ 0.5 mm Hg, *(b)* higher doses of I (5.5 to 167 μM) increased RC (from 37 $\pm$ 12% to 177 $\pm$ 39%) without change in PP. In 4 rabbits, 27 to 139 μM of I increased RC by 167 $\pm$ 27% to 612 $\pm$ 90% with no change in PP. In addition, in 7 rats, AS (55 μM) induced a 22 $\pm$ 3% decrease in RC with slight increase in PP. Higher doses of AS (550 to 1110 μM) had no significant effect on RC and PP. These results demonstrate that in the rat RR was decreased at a dose of I that is known to act primarily on PG synthesis, and RR was increased at higher doses known to inhibit phosphodiesterase (PDE) activity. AS which does not inhibit PDE activity acted similarly to a low dose of I. These findings could be further substantiated using a potent PDE inhibitor, theophylline. In conclusion, the use of NSAID to explore the role of PG synthesis inhibition alone must be undertaken with caution.

Hypolipemic effect of *d,l*-carnitine in chronic hemodialysis patients. *S. Di Giulio, B. Lacour, J. Chanard, C. Ciancioni, B. Lebkiri, M. Haguet, C. Parry, C. Basile, and T. Drüeke. Hôpital Necker, Centre E. Rist, Clinique de l'Alma, AURA, Paris and CHU Reims, France.* Hypertriglyceridaemia (HTG) in hemodialysis patients may be associated with carnitine deficiency. Carnitine is necessary for intramitochondrial transfer and oxidation of free fatty acids and thus for prevention of HTG. In the present multicentre study, 51 chronic hemodialysis patients with HTG were given a daily oral dose of 2.4 g *d,l*-carnitine during 30 days to investigate a possible hypolipaemic effect. No patients

had been on oral contraceptives, and none were alcoholics or had obesity, diabetes, or other endocrinopathies. After 30 days of *d,l*-carnitine treatment, the mean ± SEM serum triglycerid (TG) concentration decreased significantly from 3.5 ± 0.39 to 2.87 ± 0.27 mmoles/liter ($P < 0.02$) in all pts. However, serum total cholesterol did not change but HDL cholesterol increased significantly from 0.89 ± 0.05 to 1.35 ± 0.07 mmoles/liter ($P < 0.001$). When analyzing the patients in terms of treatment efficacy, we found that two groups could be distinguished: 38 patients (74%) had a decrease in serum TG concentration during *d,l*-carnitine treatment ("responders") from 3.75 ± 0.52 to 2.66 ± 0.35 mmoles/liter. However, 13 patients (26%) had no change or even an increase. The administration of *d,l*-carnitine led to a significant decrease of serum triglycerides in hemodialysis patients, possibly via the correction of carnitine deficiency.

Proteinuria and anionic sites in the glomerular basement membrane. *M. Doriaux, R. Van Holder, J. Sennesael, and P. P. Lambert. Fondation Médicale Reine Elisabeth, Bruxelles, Belgique.* Canine albumin (Sigma Chem. Co.) was cationized according to Dannon et al (P.I. > 8.5). We infused 12 to 33 mg in the left renal artery per min and per 100 g of kidney weight during 15 to 200 min. Transitory anuria in the left kidney was observed in three dogs. On the average, left GFR decreased 60% and right GFR 10%, PAH clearance decreased 64 and 24% respectively. On the left side, proteinuria (> 1 mg/min) appeared in six dogs; on the right side in three dogs. Protein excretion appeared earlier in the perfused kidney; it averaged 3.2 mg/min compared with 3.4 mg/min in the contralateral kidney. Electron microscopy showed in both kidneys the formation of clump-like deposits on both sides of the glomerular basement membrane. The clumps were more regularly distributed on the epithelial border than on the endothelial border, producing on tangential slices a lattice-like pattern comparable to that observed using other electropositive tracers. The degree of labeling of the anionic sites varied from one kidney to the other, being more pronounced when proteinuria was present. Nevertheless, a discrete clump formation was occasionally observed in the contralateral kidney when protein excretion remained below 1 mg/min. Neither clump formation nor proteinuria could be produced by intrarenal infusion of anionic canine albumin at equivalent dose.

The kidney in systemic scleroderma: A study of 38 consecutive cases. *B. Francois, G. Moulin, H. Assenat, and B. Jandot. Renal Section, Dermatology Section, Hôpital de l'Antiquaille, 69005 Lyon, France.* The renal status of 38 patients with progressive systemic scleroderma was investigated by the usual clinical tests, urine immunoelectrophoresis, PAH, and inulin or ^{125}I-iothalamate clearances and in 4 cases a renal biopsy. Fourteen patients presented with proteinuria and/or a high serum creatinine and/or hypertension, with low clearance values in all cases. In 14 other patients, an abnormality was apparent from clearance results (12 cases), renal biopsy (1), urine immunoelectrophoresis (1). The earliest sign of renal involvement that could be demonstrated was a reduced renal plasma flow as determined by the PAH clearance and an elevated filtration fraction. A glomerular mininal change electrophoretic pattern seemed to appear subsequently as either isolated (9 cases) or associated with abnormal clearance values (8 cases).

Plasma exchange therapy in steroid-resistant kidney allograft rejection. *G. Fruchaud, A. A. Cattaneo, M. Fabre, T. Nebout, A. T. Sobel and B. Weil. Department of Nephrology and Plasma Exchange Committee, Hôpital Henri Mondor, Creteil, France.* Plasma exchanges (PE) have been used as additional treatment of acute and subacute rejection crises of kidney allografts. The overall benefit of this treatment has been previously difficult to assess because of the lack of satisfactory criteria in highly heterogeneous groups of patients. In this report we tried to reevaluate PE therapy as part of the therapeutic approach of renal transplantation. The effect of PE therapy was assessed only by considering the outcome of each allograft, particularly with regard to the interval before resumption of hemodialysis (CH), when this occurred. Nine patients were given 11 sets of PE (fresh frozen plasma exclusively) with an average of 7 PE per patient. PE were performed when an acute or subacute rejection crisis occurred, that was resistant to at least 4 days of high doses prednisolone (> 2 mg/kg/day) and/or methylprednisolone boli (750 mg). The duration of transplantation before the first PE averaged 133 days (range, 8 to 456 days). Five patients recovered a fair renal function (mean creatinine, 198 μmoles/liter) that appeared stable 193 days after PE (52 to 492 days). Four patients without any improvement of renal function had to resume CH *192 days* after the crisis onset (range, 8 to 226 days). Six patients who were comparable in terms of graft duration until the rejection crisis and response to steroid treatment were not given PE. All 6 patients had to resume CH *100 days* later (range, 5 to 255 days). The data suggested that *(1)* PE may sometimes provide an alternative procedure for recovery of steroid resistant rejection crisis; *(2)* when the response is not satisfactory, PE may delay CH for as much as 3 months (financial saving ~ US $10,000 per patient).

Experimental approach to the treatment of lupus nephritis by use of an accelerated model of NZBxNZW mouse disease. *M. Gayral-Ta Minh, G. J. Fournié, M. Mignon-Conte, and J. J. Conté. Laboratoire d'Immunopathologie Rénale, Service de Néphrologie et d'Hémodialyse. C. H. U. Toulouse-Purpan Toulouse-Cedex, France.* The NZBxNZW mouse model has been widely used to evaluate the effects of various drugs on lupus nephritis. The aim of the present study was to develop an accelerated model of NZBxNZW mouse disease and to establish whether it was suitable for therapeutic studies. For this purpose, the effects of early immunization with DNA and of injection of bacterial lipopolysaccharide (LPS) on the glomerulonephritis of NZBxNZW mice were studied. Combined injections of DNA complexed to methylated bovine serum albumin (DNA-mBSA) and of LPS appeared to be more efficient in accelerating the disease of NZBxNZW mice than injections of DNA-mBSA or LPS alone. A rapid increase in levels of anti-DNA antibodies, an early appearance of severe renal lesions and a shortened survival were observed in mice injected with both DNA-mBSA and LPS. Mice with accelerated disease were treated with cyclophosphamide or heparin. The efficacy of cyclophosphamide for the treatment of the NZBxNZW mouse disease was shown by immunologic and histologic studies in mice younger than 4 months. Heparin appeared to have a beneficial effect by preventing the endocapillary cellular proliferation induced by injections of DNA-mBSA and LPS. The accelerated model of NZBxNZW mouse disease might be a useful tool for experiments on the treatment of lupus nephritis.

Passive-active immunization against hepatitis B in a high-risk setting. *N. Geslin, D. Pierre, P. Lebon, Ph. Maupas, P. Dubois, A. Goudeau, P. Coursaget, G. Lesage, B. Yvarnet. Hopital d'Orléans-La Source, Service d'hémodialyse, Orléans Cedex, Hôpital du Mans, Service de Néphrologie-Hémodialyse, Le Mans, Institut de virologie, Tours, France.* Since 1975, immunization against hepatitis B in man was carried out by means of a formalin-inactivated vaccine. This vaccine was proven to be safe and effective in prevention of hepatitis B infection in high-risk settings. In some volunteers, however, infections occurred before immunization was completed. Simultaneous administration of specific anti-HBs antibody and hepatitis B vaccine was performed to avoid such infections. This protocol is in fact similar to that used for the prevention of rabies and tetanus. Passive-active immunization has been applied to 11 staff members and 23 patients of a hemodialysis unit where hepatitis B was common. In 100% of ward staff members and 56% of patients, it was possible to induce active immunization under specific protection with anti-HBs immunoglobulins. This result is assessed by the

persistance and/or increase of anti-HBs while passively acquired antibodies disappeared as shown by the kinetics of anti-HBc. Primary response to the vaccine is associated with anti-HBs in the IgM class in most of the cases. Passive-active immunization remains the most effective preventive measure against hepatitis B infection in hemodialysis units for individuals who are exposed constantly to a high risk of infection.

Renal effects of ozoline, a new loop diuretic. *J. L. Imbs, M. Schmidt, and J. Schwartz. Institut de Pharmacologie, Faculté de Médecine, Strasbourg, France.* The elimination of water and electrolytes, free water clearance, renal blood flow, glomerular filtration, and renin secretion were measured for 6 hours after the i.v. injection of $6 \cdot 10^{-5}$ M/kg of (+)- or (−) -ozolinone (15.5 mg/kg) or of forosemide (20 mg/kg) in 20 pentobarbitol-anaesthetized dogs. (−)-Ozoline has the characteristics of a loop-diuretic and, at equimolar doses, is more active than furosemide. Both ozoline isomers provoke a large and prolonged rise in renal blood flow. This renal vasodilation is accompanied by a drop in GFR and in the filtered fraction, which suggests that the vasodilatory action predominantly affects the efferent glomerular arteriole. Distinguishing between hemodynamic modifications and salidiuretic response enables the effects of the two isomers to be analyzed, and favors the study of the parameters involved in renin hypersecretion provoked by loop diuretics. Only (−)-ozoline, a salidiuretic and vasodilator, provokes renin hypersecretion like that induced by furosemide. (+)-Ozoline, which is only a vasodilator, does not significantly modify renin secretion.

Influence of oral contraceptive therapy on lupus nephritis activity. *P. Jungers, M. Dougados, C. Pelissier, F. Kuttenn, F. Tron, Ph. Lesavre, and J. F. Bach. Inserm U 25, Departments of Nephrology and Endocrinology, Necker Hospital, Paris, France.* The deleterious effects of estrogens in experimental murine lupus are well established. We retrospectively studied the relationship between oral contraceptive (OC) therapy and the activity of lupus disease in 36 females with severe forms of systemic lupus erythematosus (SLE), observed at Necker Hospital from 1974 to 1979. All patients had renal involvement, documented by renal biopsy in 32, of which 9 showed diffuse proliferative glomerulonephritis. Immunologic assessment of SLE (anti-DNA binding, serum complement, PEG test, cryoglobulinaemia) was performed every 3 months during the study period. Combined preparations containing either 50 μg (14 pts) or 30 μg (7 pts) of ethinyl-estradiol were given to 21 women. Initial signs of SLE developed in 5 of them, and exacerbation of quiescent SLE in 6 others, during the 3 to 24 months following the start of OC therapy, an overall flare-up incidence of 52%. Pure progestogen preparations were given as OC therapy to 11 pts, using continuous microdose progestins (6 pts), low-dose norsteroids (3 pts) or chlormadinone acetate (2 pts). With a followup of 6 to 27 months, none of these patients developed clinical or immunologic signs of SLE exacerbation. Similarly, sequential substitutive therapy with percutaneous 17 beta-estradiol and chlormadinone acetate was used in 4 women with persistent amenorrhoea following cyclophosphamide therapy, without any sign of exacerbation of quiescent SLE (followup: 18 to 29 months). We conclude that oral contraceptive therapy using estrogens, even at low dosage, often induces exacerbation in SLE patients. In our experience, pure progestins were effective and devoid of such deleterious effects, and should be preferred in these patients.

Plasma and urinary oxalic acid in normal subjects, in patients with chronic renal failure and in patients treated by maintenance hemodialysis. *A. Krouri, C. Jacobs, G. Charransol, and J. P. Clavel. Groupe Hospitalier Pitié-Salpétrière, Paris, France.* The mechanisms of renal excretion of oxalic acid (OA) remain controversial. Depending on the method used for measuring plasma and urinary OA, renal clearance of OA is evaluated in normal subjects between 10% and 200% of GFR. In this study, plasma OA (measured by colorimetric method) and urinary OA (measured by gas-chromatography) are determined in 34 control subjects (17 males, 17 females) with normal GFR (creatinine clearance, 109 ± 14 ml/mn), in 3 groups of 8 patients each with CRF whose average C_{Cr} are 34.6 ± 9.6 (group 1), 15.2 ± 2.4 (group 2), and 8.04 ± 1.8 min (group 3) and in 16 patients treated by maintenance hemodialysis. In the control-subjects, mean plasma level of OA is 28.4 ± 8 μmoles/liter (2.56 ± 0.72 mg); 24 hours urinary OA, 324 ± 67 μmoles/liter (29.2 ± 6 mg); and mean renal OA clearance, 9.06 ± 3.3 ml/min. A significant rise of plasma OA (49 ± 6.3 μmoles/liter, $P < 0.001$), a significant decrease of 24 hours urinary OA (262 ± 81 μmoles/liter, $P < 0.05$), and of renal clearance of OA (4.14 ± 1.2 ml/min, $P < 0.001$) compared to the values found in the control subjects is recorded only in patients with an advanced stage of CRF (group 3). In 8 patients treated by maintenance hemodialysis 15 hours per week with cuprophane membrane dialyzers and a single-pass dialysis fluid delivery system, mean plasma OA is found at 77 ± 17 μmoles/liter before dialysis, 39 ± 5 μmoles/liter after dialysis, the extraction rate of OA during dialysis being 50.8%. In 8 patients dialyzed 9 to 12 hours per week with PAN membrane dialyzers and a closed-loop batch dialysis fluid delivery system, plasma OA is found at 102 ± 32 before dialysis and 51 ± 19 μmoles/liter after dialysis, the extraction rate during dialysis being 49.8%. The differences of the values recorded in the two groups of dialyzed patients are not statistically significant. These results are at variance with those reported in the literature by the authors who use isotopic methods with ^{14}C oxalate to evaluate urinary and plasma OA. Isotopic methods yield much lower values for plasma OA, the renal clearance of OA being thereby found higher than GFR. The steady elevation of the fractional excretion of OA (OA clearance ÷ creatinine clearance) until the advanced stage of CRF is attributed to a sharp decrease of the reabsorption of OA in the proximal convulated tubule, rather than to an increase of tubular secretion.

Influence of sodium and potassium intake on renal adaptation to a chronic acid load. *J. J. Lafontaine, E. Munoz, J. P. Dieu, and C. van Ypersele de Strihou. Université Catholique de Louvain, Cliniques Universitaires St-Luc, Service de Néphrologie, Bruxelles, Belgium.* Renal response to a chronic acid load (CAL) is not modified by the presence or absence of dietary sodium chloride, an observation arguing against the concept that sodium availability in distal tubule (DT) is a determinant of renal acid excretion. Sodium chloride might have, however, a twofold effect: increased sodium availability in DT and extracellular fluid expansion decreasing DT avidity for sodium. In the present study, we have prevented extracellular fluid expansion by providing sodium and hydrogen ions with a nonreabsorbable anion sulfate. Simultaneously, the role of potassium was assessed by evaluating the influence of sodium with and without concomitant potassium depletion. Twenty-one dogs, potassium depleted with an electrolyte-free diet and Kayexalate, are given a CAL (3.5 mm/kg/day) of ammonium sulfate. In group A (12 dogs), no sodium is given; in group B (9 dogs) sodium sulfate (1.25 mg/kg/day) is added to the diet. After CAL (period I), plasma bicarbonate is lower in group A than it is in group B (14.8 vs. 19.7; $P < 0.01$). Sodium intake is thus a critical determinant of the response to CAL. Correction of the potassium deficit by the addition of potassium sulfate (1.25 mg/kg/day) (period II) increases plasma bicarbonate in both groups (+ 2.4 and 3.8 mEq/liter), confirming thus the existence of potassium deficiency acidosis. Provision of sodium sulfate to 4 potassium-repleted animals (group A, period III) increases further plasma bicarbonate (+ 8.1 mEq/liter), demonstrating that the renal response to CAL remains sodium dependent even after potassium repletion. We conclude *(1)* distal tubular sodium is a critical determinant of the renal adaptation to CAL independently of potassium balance *(2)* potassium deficiency acidosis persists even when the acid load is

given with a nonreabsorbable anion. It does not result from an increased tubular permeability to chloride.

Tuberous sclerosis with chronic renal failure treated by hemodialysis and transplantation. *A. Meyrier, M. Rainfray, and J. Roland. Service de Néphrologie, Hôpital, Tenon, Paris, France.* We report on a case of tuberous sclerosis in a 36-year-old female with chronic renal failure, who was treated by hemodialysis and transplantation. The diagnosis of tuberous sclerosis was ascertained by the association of typical cutaneous, retinal, and osseous lesions. On the contrary, the renal changes were unusual by their revealing symptoms, evolution, and pathology. Early hypertension was exacerbated in the course of two pregnancies but was never severe. It preceded the late onset of renal failure, which progressed slowly. The kidneys were small, with a peculiar angiographic aspect: the renal tissue was not invaded by hamartomas but the whole arterial bed was diffusely modified, with a cortical perfusion defect. Both kidneys were studied after bilateral nephrectomy. The lesions were far more complex than a simple replacement of the renal tissue by angiomyolipomas: they involved the vessels, the interstitium (with lipid inclusions surrounded by a macrophagic reaction), and the glomeruli (with focal and segmental sclerosis). This observation (which is the 11th case in the literature of tuberous sclerosis with chronic renal failure and the 3rd treated by transplantation) documents a particular variety of congenital and familial nephropathy with delayed appearance.

Nephropathy and hepatic peliosis: Visceral light chain deposition in a pleomorphic lymphoplasmacytic malignancy analogous to Waldenström's macroglobulinemia. *F. Mignon, M. Cerf, J. L. Preud' Homme, J. C. Brouet, and L. Morel-Maroger. Service de Néphrologie et I.N.S.E.R.M. U 64, Hôpital Tenon et I.N.S.E.R.M. U 108 Hôpital Saint Louis, Paris, France.* A 38-year-old woman was in good health until she developed portal hypertension in May of 1978. Liver biopsy showed sinusoidal ectasia and widening of Disse spaces consistent with peliosis hepatis. In addition, there were amorphous, nonamyloid deposits along the sinusoids and in the Disse spaces, which exhibited a very dim staining for anti-kappa serum. After cavography, the patient underwent reversible anuria. Later, proteinuria without hematuria and renal failure was detected, and renal biopsy showed widespread glomerular and tubular deposits with a strong fixation of the anti-kappa conjugate along the tubular basement membranes. These findings led to search for a lymphoid malignancy. Marrow biopsy showed a pleomorphic lymphoplasmocytic proliferation analogous to that of Waldenström's macroglobulinemia. But no serum and urine monoclonal Ig could be detected on repeated examinations. Biosynthesis experiments allowed the conclusion that the cells synthesized normal size Sig which were virtually nonsecreted, and abnormally short κ chains. There was a considerable difference between the apparent modecular weights of the short light chains in the cytoplasmic and excreted chains. The patient died rapidly with hepatic failure and hypercalcemia. In conclusion, our patient presented a lymphoplasmacytic disorder, revealed by visceral deposits. Randall et al have described this new syndrome, called systemic light chain deposits. In our case, biosynthesis experiments suggest that the visceral lesions may be related to the deposit of structurally altered immunoglobulin chains, synthesized by plasma cells. In the liver, the deposits may be the cause of the hepatic peliosis, because various injuries to the sinusoidal lining cells are apparently able to induce the occurrence of this anatomic entity.

Correlations between liver biopsy data and HB virus antigenemia in 108 patients treated by chronic hemodialysis. *C. Naret, P. Jungers, C. Degott, F. Degos, A. M. Couroucé, H. Kreis, and J. Crosnier. INSERM U 25 and Department of Nephrology, Necker Hospital, INSERM U 24 and Department of Hepatology, Beaujon Hospital, and Centre National de Transfusion Sanguine, Paris, France.* From 1969 to 1979, 108 uremic patients treated by maintenance hemodialysis (62 males, 46 females) underwent liver biopsy (LB) either because of clinical evidence of chronic active hepatitis (59 pts) or routinely during abdominal surgery (23 pts) or kidney transplantation (26 pts). Repeated LB were performed in 31 pts (in 19, 1 to 5 yrs after transplantation). Liver biopsies showed chronic active hepatitis (CAH) in 20 pts (18.9%), chronic persistent hepatitis (CPH) in 36, lobular hepatitis (LH) in 15, and normal liver in 37. All 20 pts with CAH (12 males, 8 females) were chronic HBsAg carriers, with 80% of them positive for HBeAg. None of the patients with transient or without antigenemia was found to have CAH. Such lesions were found in only 4 out of the 49 patients with routine LB. HLA B8 frequency was 15.3% in CAH, 16.6% in CPH, 18.8% in LH, and 24.2% in normal liver. Mean interval between initiation of dialysis treatment and LB was not longer in CAH (25.1 $\pm$ 2.8 months) than in CPH patients (36.3 $\pm$ 3.7 months). Iterative LB in 12 dialysis patients showed an aggravation of lesions only in 4 of the 8 persistently positive for HBsAg. Repeated LB in 19 transplanted patients showed aggravation in 10, all positive for HBsAg. Progression to liver cirrhosis occurred in 6 patients, with fatal outcome in 4, an overall mortality of 4%. We conclude that in uremic hemodialyzed patients CAH lesions are found only in chronic carriers of HBsAg. Although further progression of liver lesions may occur during dialysis treatment and after renal transplantation, fatal evolution is rarely observed.

Diversity of human T lymphocyte differentiation antigens. *P. Niaudet. I.N.S.E.R.M. U.192, Hôpital des Enfants-Malades, Paris, France.* To identify cell surface proteins on human T lymphocytes, we prepared two different heteroantisera. Cell surface proteins were iodinated or internally labeled with amino acids. After cell solubilization and immunoprecipitation with specific antisera, the proteins recognized were analyzed on polyacrylamide gels by autoradiography or fluorography. The rabbit antimonkey thymocyte antisera precipitated a single glycoprotein of 33,000 daltons and one or several polypeptides of 40 to 45,000 daltons. The rabbit anti-Sezary cell antisera reacted with different proteins (55,000, 65,000, 90,000, 170,000 daltons). Furthermore, we have shown that these various proteins are expressed differently in various T cell types (thymocytes, T-leukemia, peripheral T cells). Thus, these proteins define different T lymphocyte populations according to the stage of maturation. The use of specific antisera that define functional subsets is of great interest. For example it may allow identification of the cells responsible for transplant rejection and to better analyze autoimmune diseases. These results could have therapeutic incidence.

Vaccine against hepatitis B in children: Prevention of hepatitis in a pediatric hemodialysis unit. *H. Nivet, J. Drucker, Ph. Maupas, F. Dubois, P. Coursaget, A. Goudeau, J. C. Rolland, J. Pengloan, Ph. Bagros, and B. Grenier. Hôpital G. de Clocheville, Tours, France.* In a pediatric hemodialysis unit, 8 children (aged 2 to 14 years) with chronic renal failure were immunized against hepatitis B infection by means of a vaccine. The vaccine was prepared by purification of HBsAg from human sera and was formalin inactivated. Six of them were undergoing periodic hemodialysis, and two were still managed with conservative treatment. Three HBsAg chronic carriers dialyzed in the unit represented a permanent risk of hepatitis B infection for the whole unit. All the vaccinated children sero-converted for anti-HBsAg after vaccination. A followup from 3 to 17 months did not show clinical, biologic, or serologic signs of hepatitis B in the vaccinated children. Therefore, hepatitis B vaccine in children with chronic renal failure appeared to be safe and its potency satisfactory. Presence of anti-HBs and absence of appearance of hepatitis B following immunization suggested that hepatitis B vaccine represented for these children an efficient means of prevention against hepatitis B infection.

Vaccination against hepatitis B: A 4-year followup in an adult hemodialysis unit. *J. Pengloan, H. Nivet, M. Perrier, Ph. Bagros, Ph. Maupas, A. Goudeau, P. Coursaget, and F. Dubois. Unité d'Hémodialyse and Unité de Virologie, Service Hémodialyse, CHU Bretonneau, Bd Tonnellé, Tours, France.* Since October 1975, 71 staff members and 92 haemodialysis patients were immunized against hepatitis B in an adult haemodialysis unit. The vaccine was prepared by purification of HBsAg from human sera and was formalin inactivated. Vaccinees were screened for markers of HB virus infection, i.e., HBsAg, anti-HBs, anti-HBc; serial controls of serum transaminases (A.L.T., A.S.T.) and of markers of auto-immunity were performed. The vaccinated patients were followed from 6 to 50 months. No evidence of autoimmunity was observed. Of staff members, 96% sero-converted for anti-HBs; none of them evidenced clinical, biologic, or serologic, signs of active HB infection. Of the hemodialysis patients, 65.6% sero-converted for anti-HBs; none of them became HBsAg chronic. These results were obtained despite the fact that nearly 30% of the patients present in the unit at the time of the trial were HBsAg chronic carriers. Active immunization proved to be a safe and efficient method to prevent HB infection in a hemodialysis unit.

Plasmapheresis in systemic lupus erythematosus: Effects on immunologic pamameters. *J. P. Pourrat, J. Lule, J. P. Calot, G. J. Fournié, and J. J. Conté. Laboratoire d'Immunopathologie Rénale, Service de Néphrologie et d'Hémodialyse. C.H.U. Toulouse-Purpan, Toulouse, Cedex, France.* Twelve patients with systemic lupus erythematosus were treated by plasma exchanges. All had histologic evidence of lupus nephritis. The plasma exchanges were always performed during an acute phase of the disease (renal or/and extra-renal) with classical immunologic disorders (presence of anti-DNA antibodies and of circulating immune complexes). The effects of the plasma exchanges on serum immune complexes, anti-DNA antibodies, and complement components were studied during and after the plasma exchanges. Circulating immune complexes (determined by the fluid phase Clq-binding test) that were found in all patients before the exchanges, disappeared in most cases after ten daily exchanges of 4 liters. After the plasma exchanges, various kinetics of variations of circulating immune complexes were observed. The levels of anti-DNA antibodies were modified to a lesser extent: high levels may remain after exchanges up to 40 liters. After several weeks, anti-DNA antibody levels were frequently as high as they were before plasmapheresis in the absence of additional immunosuppressive treatment. The complement component levels were directly lowered by the plasma exchanges, and their study appeared of limited interest. A clinical improvement was generally observed, but in the absence of a control group of patients, no definite conclusion on the clinical improvement after plasmapheresis can be drawn from this study. The place of plasmapheresis in the treatment of patients suffering from systemic lupus erythematosus remains to be more precisely defined.

Proximal tubular siderosis in paroxysmal nocturnal hemoglobinuria. *P. Simon, M. P. Ramée, A. Gouazic, M. Ben-Zenou. Centre Hospitalier, Saint-Brieuc, Pontchaillou, Rennes, France.* A patient with paroxysmal nocturnal hemoglobinuria, who required regular blood transfusions for hemolytic episodes, had an acute renal insufficiency after ingestion of glafenine (1.50 g during a single day). The renal function improved spontaneously in 10 days. Renal biopsy tissue was rust colored and, on microscopic examination, showed marked proximal tubular siderosis as well as mononuclear interstial infiltrates. Immunofluorescence was negative for immunoglobulin G, A, M, C_3, and fibrin. After 1 year, because tubular proteinuria appeared without other functional abnormalities, a new renal histologic examination was made and showed identical lesions. Marked deposition of iron in renal proximal tubules is typical of paroxysmal nocturnal hemoglobinuria.

Repetitive hemodialysis by temporary venous subclavian cannulation. *O. Simons, F. Khazine, T. Haas, G. Dongradi, and J. P. Fendler. Centre Hospitalier Intercommunal de Poissy, Poissy, France.* Since January 1979, temporary access of a subclavian vein has been used instead of venous femoral cannulation (used from 1974 to 1978) and instead of Scribner's shunt (used before 1974). In 22 chronic renal failure patients without permanent vascular access, a subclavian cannula is introduced by the Seldinger technique via a subclavicular approach, and its position in the superior vena cava is checked by fluoroscopic control. Through an Y connection, extracorporeal circulation is achieved by a single-needle device with double-head pump without loss of dialysis efficiency. After each session, the cannula is flushed with heparinized saline and kept in place under sterile adhesive dressing. The average duration of insertion was 8 weeks and the longest 8 months and 1 week. The immediate complications were: puncture of an arterial vessel, 3 (hemothorax, 1; hemomediastinum, 1; puncture without complication, 1); pneumothorax, 0. Secondary complications were: blood stream infection, 0; thrombosis of the subclavian vein, 0; clotting or kinking of the cannula necessitating a change by reinsertion of the guide wire without the need of a separate puncture, 9; removal of the cannula necessitating a separate puncture, 2. The advantages of the temporary subclavian access are: immediate use, sufficient blood flow rate, unlimited duration of use, no need for hospitalisation, possibility of re-use of this access in case of new shunt problems, and preservation of blood vessels. This access must always be preferred to the Scribner's shunt that we have not used since 1974; it must be preferred to the femoral access when more than two dialysis sessions are needed. To keep insertion complication to a minimum, the cannula should be inserted by trained medical staff with a reliable technique.

Nephrotic syndrome following porta-caval shunt in cirrhosis: A study of three cases. *G. Touchard, D. Picaud, J. P. Saint Andre, Ph. Collin, O. Pourrat, Ph. Babin, and D. Patte. Service d'Anatomie Pathologique et Service de Néphrologie, CHU. Hotel Dieu, Poitiers, France.* Three cirrhotic male patients with nephrotic syndrome after undergoing surgery for porta-caval shunt have been studied. They were 42, 48, and 56 years old, respectively, and did not have proteinuria before the operation. In two patients, the serum gammaglobulin level increased after porta-caval shunt, and renal manifestations appeared 6 months after the operation. In the third patient, the nephrotic syndrome appeared 4 years after shunt. Nonselective proteinuria, microscopic hematuria, and moderate renal failure were constant. Antinuclear antibodies, HBsAg, and cryoglobulin were absent. In two patients, a low C3 level was found. Renal biopsy showed membranoproliferative glomerulonephritis in three patients, with diffuse epithelial crescents in one. Mesangial and subendothelial granular IgA deposits were found in two patients. The clinical course was death from septicemia in one; hypertension, terminal renal failure and hemodialysis in the second; spontaneous disappearance of the nephrotic syndrome with persistent mild proteinuria and renal failure in the third. These three patients suggest that porta-caval shunt may play a significant pathogenetic role in hepatic glomerulonephritis.

Carpal tunnel syndrome in hemodialysis patients. *J. Zingraff, S. Di Giulio, S. Hafez, J. Benoit, D. Kuntz, P. Jungers, and J. L. Funck-Brentano. Inserm U 25, and U 90 and Department of Nephrology Necker Hospital, Department of Orthopedic Surgery, Ambroise Paré Hospital and Viggo-Petersen Center, Paris, France.* Carpal tunnel syndrome (CTS) has been reported in patients receiving regular dialysis treatment (RDT), but it was not considered to be a common complication in this treatment. We observed CTS in 16 hemodialysis patients (6 males, 10 females) out of more than 1000 patients who had started RDT at Necker Hospital and associated centers between 1964 and 1979, an overall incidence of near 2%. The mean interval between initi-

ation of RDT and clinical evidence of CTS was 8 years (range, 4 to 12 years). CTS was bilateral in nine patients. It was never observed in arms or legs untouched by vascular access procedures. Temporary relief of symptoms (pain and/or paresthesias) was observed after local corticosteroids infiltration. This relief, together with nerve conduction velocity studies, permits the differentiation of CTS from vascular steal syndrome and from acroparesthesias and shoulder pain of osteoarticular origin. The carpal tunnel was surgically decompressed in 11 patients. Complete recovery was obtained in all but one, suffering from synovial amyloidosis. In conclusion, with the increasing number of patients on maintenance hemodialysis for more than 4 years, diagnosis of CTS should be considered in any dialysis patient suffering from hitherto unexplained pain in the upper limbs. In our experience, vascular access procedures always preceded development of the syndrome.

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Effects of thromboxane synthetase inhibition and thromboxane B_2 administration on glomerular function in the rat. *C. Baylis. Department of Physiology, Manchester University, Manchester, England.* The normal rat kidney possesses the enzymes necessary to synthesize the potent vasoconstrictor and proplatelet aggregating agent thromboxane A_2 (TXA_2), and its stable and less potent metabolite thromboxane B_2 (TXB_2). It is not at present known, however, whether thromboxane (TX) synthesis is activated in the normal kidney, or what effect TX has on renal function in vivo. The following experiments were performed to investigate these questions. Micropuncture experiments were carried out on 7 adult male Munich-Wistar rats (a strain that possesses glomeruli on the kidney surface) in control conditions and again during i.v. infusion of imidazole (0.05 mmoles/kg/min). In vitro studies have indicated that imidazole is a potent TX-synthetase inhibitor. No significant change was seen in single nephron (SN) GFR or in single glomerular plasma flow rate (Q_A) with imidazole. Protein concentration (C) in afferent glomerular blood (C_A) fell slightly, but efferent glomerular protein concentration (C_E) was not significantly affected by the drug. The mean hydrostatic pressure difference across the glomerular capillary wall, $\bar{\Delta}P$, rose markedly from 35 ± 2 to 45 ± 1 mm Hg ($P < 0.25$, paired t test), due largely to increased glomerular capillary hydrostatic pressure (P_{GC}). In control conditions, rats were at filtration pressure equilibrium (i.e. $\bar{\Delta}P \equiv$ oncotic pressure of efferent glomerular blood, Π_E), and only minimum values of the glomerular capillary ultrafiltration coefficient (K_f) could be calculated. Filtration pressure disequilibrium ($\bar{\Delta}P > \Pi_E$) obtained during imidazole infusion and exact values of K_f could be calculated. K_f, the product of glomerular water permeability and available filtration surface area, averaged a minimum of 0.105 ± 0.01 nl/(sec·mm Hg) in control conditions and fell significantly to 0.057 ± 0.005 nl/(sec·mm Hg) during imidazole infusion ($P < 0.005$). Mean femoral arterial blood pressure ($\overline{AP}$) also rose significantly with imidazole from 113 ± 4 to 131 ± 4 mm Hg ($P < 0.001$). Inhibition of a potent vasoconstrictor system would be expected to increase Q_A and SNGFR; thus, the lack of change in these variables during TX-synthetase inhibition with imidazole may suggest that little or no TX is synthesized under normal conditions in the rat kidney. A *selective* inhibitor of vasoconstrictor TX would however reduce, rather than raise $\overline{AP}$, and since *increases* in blood pressure were seen with imidazole it seems likely that this drug exerts additional actions in vivo. Similar experiments were carried out on 6 additional rats in which TXB_2 (100 μg/kg/min) was infused into the aorta above the left renal artery. A significant fall was seen in SNGFR with TXB_2 (from 34.7 ± 4.7 to 26.4 ± 4.2 nl/min, $P < 0.005$) and Q_A also declined (from 111.6 ± 16.1 to 91.2 ± 12.6 nl/min, $P = 0.05$, $N = 5$). A small but significant increase occurred in C_A (from 5.7 ± 0.2 to 6.2 ± 0.2 g/dl, $P < 0.005$) while C_E did not change. The value of $\bar{\Delta}P$ was unaffected by TXB_2, and although a slight increase in $\overline{AP}$ was seen, this was not statistically significant. Rats remained at filtration pressure equilibrium during TXB_2 infusion, and thus exact values of K_f could not be calculated. The decline in SNGFR seen during TXB_2 infusion was due both to falls in Q_A and increases in C_A, the latter increasing the oncotic opposition to the ultrafiltration process. Thus, it seems that TXB_2 exerts mild renal vasoconstrictor effects at the large doses that were administered in these experiments.

Role of the major histocompatibility system in idiopathic membranous nephropathy: Prediction of outcome and response to treatment. *S. A. Cairns, P. Klouda, P. A. Dyer, J. Manos, N. P. Mallick, and R. Harris. Department of Renal Medicine, Manchester Royal Infirmary, and Department of Medical Genetics, St. Mary's Hospital, Manchester, England.* We have demonstrated a relationship between idiopathic membranous nephropathy (IMN), the major histocompatibility system (MHS) antigen HLA-DRW3, and complement factor B allotype BfFl. Like the MHS, factor B is coded on chromosome 6, and, in our series delta values suggest that BfFl, DRW3, and B18 exist as a haplotype. The clinical course of 40 patients with IMN, followed for between 2 and 23 years, has been studied in relation to immunogenetic markers (Table). The clinical presentation was similar in the three groups and there was no difference in incidence of familial renal disease or immunochemical abnormalities. Patients in group I had a worse prognosis than those in groups II and III; none remitted, while over 50% of those in groups II and III did ($P = 0.009$, Fisher's exact test). Interestingly, one third of those remitting subsequently relapsed. Decline in renal function occurred in 71% of group I patients but occurred less frequently in groups II (22%) and III (33%) ($P = 0.026$).

Group	No. of patients	B18	BfFl	DRW-3
I	7	+	+	+
II	22	–	–	+
	1	+	–	+
	1	–	+	+
III	7	–	–	–
	2	+	–	–

Eighteen of the patients received steroids. Improvement in renal function and/or decline in proteinuria occurred in 6, 5 of whom were in group II (DRW3 positive, BfFl negative). None of 3 patients treated in group I responded. IMN is immunogenetically heterogeneous, and immunogenetic markers may prove valuable in determining disease outcome. The suggestion from our data that patients responsive to treatment may be identified by MHS